Exhibit 26

Expert Report of Kristin Andruska, MD, PhD

Rothchild v. United States
No. 7:23-cv-00858-D-KS

February 6, 2025

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socioeconomic status, disease burden is less.[64,65,66] However, the opposite is true in Parkinson disease. Higher socioeconomic status is linked to higher rates of Parkinson disease, and toxic exposures are linked to the increasing burden of PD.

Numerous by-products of the Industrial Revolution, including specific pesticides, solvents, and heavy metals, have been linked to Parkinson disease.[67,68] Countries that have undergone the most rapid industrialization have seen the greatest increase in the rates of Parkinson disease.[69] Much of the original data supporting these associations were determined in the Parkinson's Environment and Genes (PEG) study, which evaluated hundreds of people with Parkinson disease, compared to control subjects, and determined positive odds ratios for environmental exposures and PD.[70,71]

Exposures that can trigger Parkinson disease can include medications, poisoning, infections, injury, and toxins, including TCE and PCE. A causative link between PD and toxin exposure was first discovered in the 1982 in northern California when a cohort of seven unrelated individuals presented with acute parkinsonism, after injecting synthetic drugs later discovered to be contaminated with the toxin 1-methyl-4-phenyl-1,2,3,6-

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[64] Marmot M. The influence of income on health: views of an epidemiologist. *Health Aff (Millwood)*. 2002;21(2):31-46.

[65] Bloom DE, Canning D. Policy forum: public health. The health and wealth of nations. *Science*. 2000;287(5456):1207-1209.

[66] GBD 2015 SDG Collaborators. Measuring the health-related Sustainable Development Goals in 188 countries: a baseline analysis from the Global Burden of Disease Study 2015 [published correction appears in Lancet. 2017 Jan 7;389(10064):e1.

[67] Goldman, Samuel M. “Environmental toxins and Parkinson's disease.” Annual review of pharmacology and toxicology vol. 54 (2014): 141-64.

[68] Tanner, Caroline M et al. “The disease intersection of susceptibility and exposure: chemical exposures and neurodegenerative disease risk.” Alzheimer's & dementia : the journal of the Alzheimer's Association vol. 10,3 Suppl (2014): S213-25.

[69] GBD 2016 Parkinson's Disease Collaborators. Global, regional, and national burden of Parkinson's disease, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016 [published correction appears in Lancet Neurol. 2021 Dec;20(12):e7.

[70] Gatto NM, Cockburn M, Bronstein J, Manthripragada AD, Ritz B. Well-water consumption and Parkinson's disease in rural California. Environ Health Perspect. 2009;117(12):1912-1918.

[71] Adler T. Pesticides and Parkinson's disease: the legacy of contaminated well water. Environ Health Perspect. 2009;117(12):A553. doi:10.1289/ehp.117-a553a

tetrahydropyridine (MPTP).[72] This cohort of patients and their MPTP-induced parkinsonism has been studied longitudinally and has had a profound impact on our understanding of PD over the last 30 years. We continue to use MPTP-induced animal models of Parkinson disease to study its scientific basis and treatments,[73] and I personally care for the last remaining survivor of the MPTP Seven.

Since the discovery of MPTP's neurotoxic effects, environmental compounds structurally similar to MPTP have been linked to cell death of dopaminergic neurons and Parkinson disease. Once MPTP enters the brain, it metabolizes into MPP+ the molecule that causes neurotoxic damage and dopaminergic cell death. MPTP's toxic metabolite MPP+ is also called cyperquat, an herbicide that has previously been banned Another related chemical, the herbicide paraquat, has been linked to an increased risk of PD and banned in 70 countries.[74]

Of note, the compound TaClo (1-trichloromethyl-1,2,3,4-tetrahydro-β-carboline) is structurally very similar to MPTP. Both easily penetrate the blood–brain barrier and exert distinct toxicity on dopaminergic neurons. Importantly, both TaClo and MPTP enter the brain and block tyrosine hydroxylase, the enzyme responsible for making dopamine. Their common effect of dopamine blockade accounts for their shared toxic phenotype of Parkinson disease. As repeatedly demonstrated in animal models[75,76,77,78]

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[72] Langston, J W et al. "Chronic Parkinsonism in humans due to a product of meperidine-analog synthesis." *Science (New York, N.Y.)* vol. 219,4587 (1983): 979-80.

[73] Langston JW. The MPTP Story. *J Parkinsons Dis*. 2017;7(s1):S11-S19.

[74] Chiba K, Trevor A, Castagnoli N Jr. Metabolism of the neurotoxic tertiary amine, MPTP, by brain monoamine oxidase. *Biochem Biophys Res Commun*. 1984;120(2):574-578.

[75] Bringmann G, Feineis D, God R, et al. 1-Trichloromethyl-1,2,3,4-tetrahydro-beta-carboline (TaClo) and related derivatives: chemistry and biochemical effects on catecholamine biosynthesis. *Bioorg Med Chem*. 2002;10(7):2207-2214.

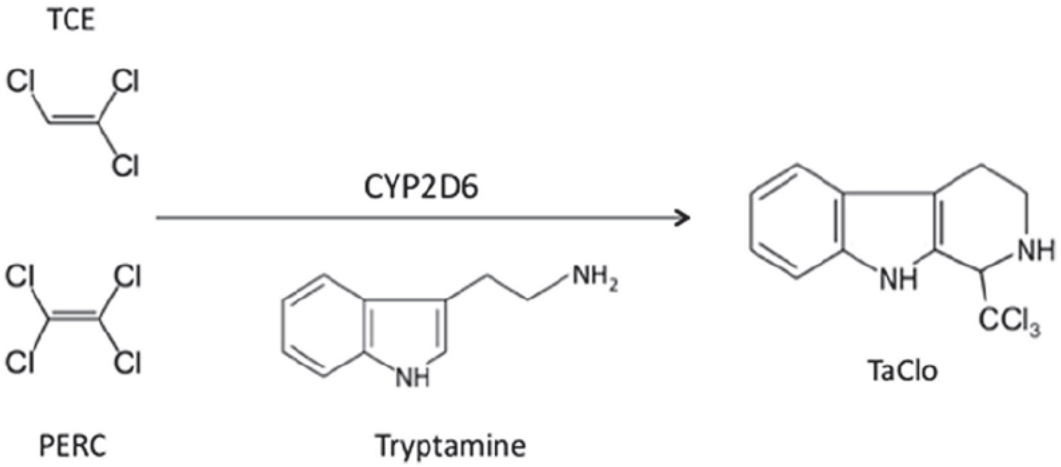
[76] Grote C, Clement HW, Wesemann W, et al. Biochemical lesions of the nigrostriatal system by TaClo (1-trichloromethyl-1,2,3,4-tetrahydro-beta-carboline) and derivatives. *J Neural Transm Suppl*. 1995;46:275-281.

[77] Bringmann, G. *et al.* (1998). Highly Halogenated Tetrahydro-β-Carbolines as a New Class of Dopaminergic Neurotoxins. In: Moser, A. (eds) Pharmacology of Endogenous Neurotoxins. Birkhäuser, Boston, MA.

[78] Keane PC, Hanson PS, Patterson L, et al. Trichloroethylene and its metabolite TaClo lead to degeneration of substantia nigra dopaminergic neurones: Effects in wild type and human A30P mutant α-synuclein mice. *Neurosci Lett*. 2019;711:134437.

and primary cell cultures[79,80] TaClo damages the nigrostriatal system and is toxic to dopamine neurons, inducing a progressive neurodegeneration in a dose-dependent fashion.

TCE and PCE have similar metabolic pathways and shared downstream metabolites. This has particular relevance to PCE/TCE exposures and Parkinson's pathology, as PCE and TCE oxidize to TaClo in a reaction catalyzed by P450 enzymes (e.g. CYP2E1) located in the basal ganglia and substantia nigra (Fig. 2).[81,82,83]



[79] Rausch WD, Abdel-mohsen M, Koutsilieri E, Chan WW, Bringmann G. Studies of the potentially endogenous toxin TaClo (1-trichloromethyl-1,2,3,4-tetrahydro-beta-carboline) in neuronal and glial cell cultures. *J Neural Transm Suppl*. 1995;46:255-263.

[80] Janetzky, B. et al. Effect of highly halogenated β-carbolines on dopaminergic cells in culture and on mitochondrial respiration. Drug Dev. Res. 1999 (46):51.

[81] Tanner CM, Goldman SM, Ross GW, Grate SJ. The disease intersection of susceptibility and exposure: chemical exposures and neurodegenerative disease risk. *Alzheimers Dement*. 2014;10(3 Suppl):S213-S225.

[82] Cichocki JA, Guyton KZ, Guha N, Chiu WA, Rusyn I, Lash LH. Target Organ Metabolism, Toxicity, and Mechanisms of Trichloroethylene and Perchloroethylene: Key Similarities, Differences, and Data Gaps. *J Pharmacol Exp Ther*. 2016;359(1):110-123.

[83] Kuban W, Daniel WA. Cytochrome P450 expression and regulation in the brain. Drug metabolism reviews. 2021;53(1):1-29.

Fig. 2. 1-trichloromethyl-1,2,3,4-tetrahydro-β-carboline (TaClo) forms in the presence of tryptamine after cytochrome P450-mediated oxidation of trichloroethylene (TCE) or tetrachloroethylene (perchloroethylene, PERC).

Not only can PCE and TCE transform into the same downstream molecule, reductive dechlorination in aerobic and anaerobic groundwater conditions degrades PCE to TCE.[84,85] This is especially important for this report, as molecules that start as PCE and transform to TCE will have the same biological relevance as TCE.

The toxicity of trichloroethylene and perchloroethylene: animal studies

Reports about the toxicity of trichloroethylene date back to 1932 in a publication of the *Journal of the American Medical Association* in which McCord describes tricholoroethylene's disease- and death-inducing thresholds in animal studies. He concludes that while trichloroethylene manufacturers may find desirable qualities in its utility, it is also a potential "disaster for exposed workmen".[86]

As summarized by the ATSDR, "Neurological effects similar to those associated with trichloroethylene exposure in humans have been reported in laboratory animals following acute or repeated inhalation or oral exposures. Short-term oral administration of trichloroethylene to rats resulted in morphological changes in the trigeminal nerve. Increased handling reactivity and increased sleep time (considered possible indicators of mood disturbances) were reported in rats repeatedly exposed to trichloroethylene. Other animal studies reported trichloroethylene-induced neuropathy, auditory impairment, visual impairment, impaired cognitive function, changes in some measures of psychomotor function, behavioral effects, cardiac arrhythmia, and neurochemical or molecular changes."[87]

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[84] Pierri, D. Actual decay of tetrachloroethene (PCE) and trichloroethene (TCE) in a highly contaminated shallow groundwater system. Environmental Advances. 2021;5:1-10.

[85] Bertolini M, Zecchin S, Cavalca L. Sequential Anaerobic/Aerobic Microbial Transformation of Chlorinated Ethenes: Use of Sustainable Approaches for Aquifer Decontamination. *Water*. 2023;15(7):1406.

[86] McCord CP (1932) Toxicity of trichloroethylene. J Am Med Assoc 99, 409–409.

[87] ATSDR Toxicological Profiles. Trichloroethylene (TCE). https://www.atsdr.cdc.gov/toxprofiles/tp19-c2.pdf. Accessed 22 December 2024.

Numerous experimental studies provide biological plausibility for this association by recapitulating key pathologic characteristics of PD in humans, including mitochondrial impairment, intraneuronal aggregation of phosphorylated α-synuclein protein, and regionally specific degeneration of nigrostriatal dopaminergic neurons. Specifically, Liu et al. demonstrated progressive and selective loss of 50% of the dopaminergic neurons in mouse substantia nigra (SN) and 50% loss of dopamine in otherwise normal mice that were treated with TCE for eight months. The mice not only demonstrated mitochondrial impairment, oxidative stress, and neuro-inflammation, but also motor impairment. These are analogous to the molecular pathways responsible for Parkinson disease.[88,89,90]

As an accompaniment to the human study of dose-dependent, occupational exposure reviewed in the following section, Gash et al. tested oral administration of TCE in animals. They demonstrated selective complex 1 mitochondrial impairment in the midbrain, striatonigral fiber degeneration, and dopamine neuron loss. As above, these are analogous to the human pathologies of Parkinson disease.[91]

Similarly, after a report of a young woman who developed Parkinson disease after occupational exposure to TCE, Guehl et al. gave mice trichloroethylene and then measured neuronal death in the substantia nigra pars compacta. Treated mice developed significant dopaminergic neuronal death (50%) compared to control mice.[92]

To augment the mechanistic links and biologic plausibility of selective dopaminergic neuron toxicity between MPP+/TaClo toxic exposures and tyrosine hydroxylase/levodopa blockade discussed above, in 2021 De Miranda et al. demonstrated that TCE exposure in rats increased brain LRRK2 kinase activity,

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[88] De Miranda BR , Greenamyre JT . Trichloroethylene, a ubiquitous environmental contaminant in the risk for Parkinson's disease. *Environ Sci Process Impacts*. 2020;22(3):543-554.

[89] Keane PC, Hanson PS, Patterson L, et al. Trichloroethylene and its metabolite TaClo lead to degeneration of substantia nigra dopaminergic neurons: Effects in wild type and human A30P mutant α-synuclein mice. *Neurosci Lett*. 2019;711:134437.

[90] Liu M, Shin EJ, Dang DK, et al. Trichloroethylene and Parkinson's Disease: Risk Assessment. *Mol Neurobiol*. 2018;55(7):6201-6214.

[91] Gash DM, Rutland K, Hudson NL, et al. Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. *Ann Neurol*. 2008;63(2):184-192.

[92] Guehl D, Bezard E, Dovero S, Boraud T, Bioulac B, Gross C. Trichloroethylene and parkinsonism: a human and experimental observation. *Eur J Neurol*. 1999;6(5):609-611.

triggered dopaminergic neuron loss, elevated oxidative stress, and increased α-synuclein accumulation.[93]

The toxicity of trichloroethylene and perchloroethylene: human studies

TCE is a six-atom (two carbons, one hydrogen, and three chlorines) solvent that is clear, colorless, volatile, nonflammable, and environmentally persistent. PCE is very closely related to TCE, having only one additional chlorine atom in place of the hydrogen atom. PCE can readily transform into TCE, and their structural analogy suggests similar toxicities. TCE and PCE can be released into the air, water, and soil. Exposure can occur through skin contact, ingestion, and inhalation. TCE and PCE enter the bloodstream and spread to bodily organs. They can rapidly cross the blood-brain barrier to exert their neurotoxic effects.[94,95]

A recent EPA ruling in December 2024 highlights neurotoxicity as "the most sensitive health effect driving the unreasonable risk of PCE" and identified "neurotoxicity as the most robust and sensitive endpoint for non-cancer adverse effects from acute inhalation and dermal exposures and as the most robust and sensitive endpoint for non-cancer adverse effects from chronic inhalation and dermal exposures for all conditions of use."[96,97] The EPA also determined that TCE poses an unreasonable risk to human

[93] De Miranda BR, Castro SL, Rocha EM, Bodle CR, Johnson KE, Greenamyre JT. The industrial solvent trichloroethylene induces LRRK2 kinase activity and dopaminergic neurodegeneration in a rat model of Parkinson's disease. *Neurobiol Dis*. 2021;153:105312.

[94] *Toxicological Profile for Trichloroethylene*. Atlanta (GA): Agency for Toxic Substances and Disease Registry (US); June 2019.

[95] *Toxicological Profile for Tetrachloroethylene*. Atlanta (GA): Agency for Toxic Substances and Disease Registry (US); June 2019.

[96] EPA. Perchloroethylene (PCE); Regulation Under the Toxic Substances Control Act (TSCA). Document ID No. EPA-HQ-OPPT-2020-0720. EPA Publication No. 89 FR 103560. December 2024. https://www.federalregister.gov/documents/2024/12/18/2024-30117/ perchloroethylene-pce-regulation-under-the-toxic-substances-control-act-tsca#h-20.

[97] EPA. Risk Evaluation for Perchloroethylene. Document ID No. EPA-HQ-OPPT-2020-0720-0035. EPA Publication No. 740-R1-8011. December 2020. https://www.regulations.gov/document/EPA-HQ-OPPT-2019-0502-0058.

health, citing neurotoxicity as a significant risk from acute and chronic inhalation and dermal TCE exposure.[98] I will review this ruling in more detail in Section VIII.

The ATSDR reports that the central nervous system is a target for trichloroethylene (TCE)'s toxic effects, including "euphoria, giddiness, lethargy, confusion, subjective symptoms of vestibular impairment (dizziness, headache, nausea), difficulty swallowing, facial effects that indicate possible trigeminal nerve damage (including sensation deficits, jaw weakness, increased blink reflex latency), dysfunction of cranial nerves other than the trigeminal nerve, memory deficits, impaired hearing, impaired visual function, mood swings, muscle weakness, tremor, decreased psychomotor function, psychotic behavior, impaired cognitive function, and loss of consciousness".[99]

Perchloroethylene (PCE or PERC) is also known as tetrachloroethylene. The ATSDR also reports "It has been clearly established that the central nervous system is a target of tetrachloroethylene toxicity in humans and animals following either inhalation or oral exposure."[100] In summary of the accidental and experimental human PCE exposure literature between 1937 and 1977: Humans with minutes to days of PCE exposure have subjectively reported headache, lightheadedness, dizziness, drowsiness, loss of coordination, mood changes, difficulty sleeping, coma, and seizures. Objective testing has highlighted coordination and balance as impaired neurologic domains after PCE exposure, and electroencephalogram (brain wave) studies have confirmed quantitative

[98] EPA. Final Revised Unreasonable Risk Determination for Trichloroethylene, January 2023. https://www.epa.gov/system/files/documents/2023-01/TCE_Final%20Revised%20RD_12-21-22-FINAL-v2.pdf.

[99] ATSDR Toxicological Profiles. Trichloroethylene (TCE). https://www.atsdr.cdc.gov/toxprofiles/tp19-c2.pdf. Accessed 22 December 2024.

[100] ATSDR Toxicological Profiles. Tetrachloroethylene (PCE). https://www.atsdr.cdc.gov/ToxProfiles/tp18-c3.pdf. Accessed 23 December 2024.

central nervous system depression in humans after PCE exposure.[101,102,103,104,105,106,107,108,109,110]

Over the past five decades, numerous case studies and case series have linked industrial solvents and TCE to parkinsonism and Parkinson

[101] Carpenter, Charles P.. "The chronic toxicity of tetrachlorethylene." *The Journal of industrial hygiene and toxicology*. 1937;19.

[102] Rowe, V. K., et al. Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. *Arch. Ind. Hyg.* 1952;5:566.

[103] Stewart, R. D., et al. Human exposure to tetrachloroethylene vapor. *Arch. Environ. Health*. 1961; 2:516.

[104] Stewart, R. D., et al. Experimental human exposure to tetrachloroethylene. *Arch. Eviron. Health*. 1970;20:224.

[105] Stewart, R. D., et al. Accidental vapor exposure to anesthetic concentrations of a solvent containing tetrachloroethylene. *Ind. Med. Surg*.1961;30:327.

[106] Patel, R., et al. Pulmonary edema and coma from perchloroethylene. *J. Amer. Med. Assoc*. 1973;223:1510.

[107] Hake CL, Stewart RD. Human exposure to tetrachloroethylene: inhalation and skin contact. *Environ Health Perspect*. 1977;21:231-238.

[108] Saland G. Accidental exposure to perchloroethylene. *N Y State J Med*. 1967;67(17):2359-2361.

[109] Morgan B. Dangers of perchlorethylene. *Br Med J*. 1969;2(5655):513.

[110] Stewart RD. Acute tetrachloroethylene intoxication. *JAMA*. 1969;208(8):1490-1492.

disease[111,112,113,114,115,116,117], in addition to myriad other neurologic conditions. More recently, Gash et al. reported 30 coworkers with chronic occupational exposure to TCE who developed parkinsonism and Parkinson disease, in proportion to their physical proximity to a TCE source at work[118].

In 2012 Goldman et al. performed a study of twins, where one twin had a confident diagnosis of Parkinson disease and the other twin did not. The authors rigorously characterized the twins' exposures to many solvents and other environmental factors through occupations and hobbies. The use of twin pairs in this study was advantageous, because it controlled for genetic and shared environmental factors. Because twins are genetically similar and share many lifestyle factors, a discordant twin-pair study design is more resistant to confounding factors than a typical case-control study. One limitation of this study was the small population size, which accounts for wide confidence intervals. However, the results showed a substantially increased risk of PD for both PCE and TCE exposed subjects, with evidence of an exposure-response relationship between duration of exposure and cumulative exposure. Specifically, the adjusted OR was 6.1 for TCE (95% CI 1.2-33, p = 0.034), and 10.5 for PCE (95% CI 0.97-113, p = 0.053). Risk was significantly increased for the combined

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[111] Guggenheim MA, Couch JR, Weinberg W. Motor dysfunction as a permanent complication of methanol ingestion. Presentation of a case with a beneficial response to levodopa treatment. *Arch Neurol*. 1971;24(6):550-554.

[112] Pezzoli G, Barbieri S, Ferrante C, Zecchinelli A, Foà V. Parkinsonism due to n-hexane exposure. *Lancet*. 1989;2(8667):874.

[113] McCrank E, Rabheru K. Four cases of progressive supranuclear palsy in patients exposed to organic solvents. *Can J Psychiatry*. 1989;34(9):934-936.

[114] Tetrud JW, Langston JW, Irwin I, Snow B. Parkinsonism caused by petroleum waste ingestion. *Neurology*. 1994;44(6):1051-1054.

[115] Uitti RJ, Snow BJ, Shinotoh H, et al. Parkinsonism induced by solvent abuse. *Ann Neurol*. 1994;35(5):616-619.

[116] Guehl D, Bezard E, Dovero S, Boraud T, Bioulac B, Gross C. Trichloroethylene and parkinsonism: a human and experimental observation. *Eur J Neurol*. 1999;6(5):609-611.

[117] Kochen W, Kohlmüller D, De Biasi P, Ramsay R. The endogenous formation of highly chlorinated tetrahydro-beta-carbolines as a possible causative mechanism in idiopathic Parkinson's disease. *Adv Exp Med Biol*. 2003;527:253-263.

[118] Gash DM, Rutland K, Hudson NL, et al. Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. *Ann Neurol*. 2008;63(2):184-192.

variable TCE or PCE exposure (OR 8.9, 95%CI 1.7-47, p = 0.01).[119] An odds ratio greater than two indicates a more than doubling of the risk of the disease incidence due to exposure. A six- to ten-fold risk increase due to individual and combined exposures is staggering.

According to multiple studies performed by the United States Government's Agency for Toxic Substances and Disease Registry (ATSDR), the drinking water at Marine Corps Base Camp Lejeune in North Carolina, during periods between 1953 and 1987, was contaminated with levels of trichloroethylene (TCE), tetrachloroethylene/perchloroethylene (PCE), and other volatile organic compounds (VOCs) that exceeded the U.S. Environmental Protection Agency's maximum contaminant levels.[120] Water was contaminated by leaking underground storage tanks, industrial spills, waste disposal sites, and a dry cleaning facility.

Given the prior studies linking PCE and TCE to Parkinson disease, in 2014, Bove et al. performed a retrospective cohort mortality study of civilian workers employed at Camp Lejeune during 1973–1985 and potentially exposed to contaminated drinking water. The Camp Lejeune (exposed) cohort was compared to a cohort of Camp Pendleton (unexposed) workers employed during the same time period. The authors found a positive association between exposure and death from several causes, including Parkinson disease, hazard ratio (HR) = 3.13, 95% CI: 0.76, 12.81. A hazard ratio is the ratio of the risk of dying at a certain time in the exposed versus the non-exposed group. A ratio of one means that the risks are the same. In this study, the risk of dying with Parkinson disease is more than tripled in civilian workers at Camp Lejeune.[121]

A 2023 population-based cohort study evaluated the Parkinson disease and prodromal Parkinson disease risk among all Marines and Navy personnel who lived at Camp Lejeune, North Carolina (exposed), versus Camp Pendleton, California (not exposed), for at least three months between 1975 and 1985, with follow-up from January 1, 1997, until February 17, 2021. This was a large, well-powered, population-based study.

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[119] Goldman SM, Quinlan PJ, Ross GW, et al. Solvent exposures and Parkinson disease risk in twins. *Ann Neurol*. 2012;71(6):776-784.

[120] Maslia ML, Aral MM, Ruckart PZ, Bove FJ. Reconstructing Historical VOC Concentrations in Drinking Water for Epidemiological Studies at a U.S. Military Base: Summary of Results. *Water (Basel)*. 2016;8(10):449.

[121] Bove FJ, Ruckart PZ, Maslia M, Larson TC. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environ Health*. 2014;13:68.

Between these cohorts the risk of PD was 70% higher in Camp Lejeune veterans (OR, 1.70; 95% CI, 1.39-2.07). Furthermore, prodromal features of Parkinson disease were significantly associated with former residents of Camp Lejeune, suggesting they may be in a phase of evolving PD pathology.[122] These are key observations in this study where the population was relatively young, in light of the long (often many decades) prodromal phase of Parkinson disease, and considering the latencies that have been reported for many Parkinson disease-associated environmental risk factors.[123,124]

In 2024 a retrospective cohort study of Parkinson disease risk study was performed after an environmental assessment detected soil contamination at a dry cleaning facility that leaked TCE, PCE, and other VOCs into the ground. In general, PCE levels in buildings with or near a dry cleaner have ranged up to 55,000 $\mu g/m^3$, 1000 times higher than the safety thresholds. The study included attorneys who worked within the predicted exposure radius (based on the extent of contamination, the likely flow of chemicals, and the presence of an underground tunnel). The results showed the prevalence of PD was higher than expected in the exposed cohort, based on age and sex.[125,126,127]

In 2024, a cohort mortality study compared the mortality rates between military personnel and civilian workers employed at Camp Lejeune or Camp Pendleton between October 1972 and December 1985. The study was performed to evaluate whether drinking contaminated water at Camp Lejeune increased the risk of specific causes of death including Parkinson disease. The authors performed 40 years of mortality follow-

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[122] Goldman SM, Weaver FM, Stroupe KT, et al. Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune. *JAMA Neurol*. 2023;80(7):673-681.

[123] Langston JW, Forno LS, Tetrud J, Reeves AG, Kaplan JA, Karluk D. Evidence of active nerve cell degeneration in the substantia nigra of humans years after 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine exposure. *Ann Neurol*. 1999;46(4):598-605.

[124] Van Laar AD, Webb KR, Keeney MT, et al. Transient exposure to rotenone causes degeneration and progressive parkinsonian motor deficits, neuroinflammation, and synucleinopathy. *NPJ Parkinsons Dis*. 2023;9(1):121.

[125] McHugh T, Loll P, Eklund B. Recent advances in vapor intrusion site investigations. *J Environ Manage*. 2017;204(Pt 2):783-792.

[126] McDermott MJ, Mazor KA, Shost SJ, Narang RS, Aldous KM, Storm JE. Tetrachloroethylene (PCE, Perc) levels in residential dry cleaner buildings in diverse communities in New York City. *Environ Health Perspect*. 2005;113(10):1336-1343.

[127] Dorsey ER, Kinel D, Pawlik ME, et al. Dry-Cleaning Chemicals and a Cluster of Parkinson's Disease and Cancer: A Retrospective Investigation. *Mov Disord*. 2024;39(3):606-613.

up, between 1979 and 2018. For military personnel with Parkinson disease, adjusted hazard ratio was ≥ 1.20 and CIR > 3. For military personnel, the aHR for Parkinson disease was 2.05 (95% CI 0.86, 4.87) and CIR > 3. For civilian workers at Camp Lejeune, the adjusted HRs for death with Parkinson disease was 1.21 (95% CI 0.72, 2.04) and CIR <3. Statistical significance testing was not used to interpret this study's results, based on expert recommendations, as p-value cutoffs are not intended to be a substitute for scientific reasoning, nor do they measure the importance of a result. Rather, the significance of results was based on the aHR, the precision of the estimate as measured by the confidence interval ratio (CIR), supporting information from other studies, and analyses of bias due to exposure misclassification. An adjusted HR ≥ 1.20 was emphasized as most meaningful for this study based on the ATSDR's published meta-analyses of studies of TCE exposed workers.[128] A CIR less than or equal to 3 determined the highest level of precision. As a civilian worker at Camp Lejeune during the years included in this study, this data is particularly relevant to Ms. Rothchild's risk of dying with Parkinson disease, as it meets both the aHR and CIR thresholds for importance. These are notable hazard ratios on their own, but are even more so in light of the relatively young age of the military cohort and the long prodromal phase of Parkinson disease. Longer follow up would quantify the full impact of toxic exposures at Camp Lejeune.[129]

In 2024, Goldman et al. studied a cohort of 172,128 marines residing at Camp Lejeune between 1975 and 1985. Focusing on those people in the cohort with a diagnosis of Parkinson disease and applying exposure estimates derived by the ASTDR, they classified individuals as exposed or unexposed to volatile organic compounds including TCE and PCE in residential water at Camp Lejeune. For the exposed group, time until psychosis, fracture, and fall in Parkinson disease were all shorter, with adjusted hazard ratios (HRs) exceeding 2: psychosis HR, 2.19 (95% confidence interval [CI]: 0.99-4.83); fracture HR, 2.44 (95% CI: 0.91-6.55); and fall HR, 2.64 (95% CI: 0.97-7.21). These data demonstrate faster Parkinson disease progression in people with prior exposure to TCE, PCE, and other volatile organic compounds. The authors note TCE and PCE may contribute to a more fulminant PD phenotype through mechanisms of increased

[128] Agency for Toxic Substances and Disease Registry (ATSDR): Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases. January 12. 2017.

[129] Bove FJ, Greek A, Gatiba R, Boehm RC, Mohnsen MM. Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study. *Environ Health*. 2024;23(1):61.

inflammation, autoimmune response, mitochondrial impairment, and abnormal alpha-synuclein.[130]

[130] Goldman SM, Weaver FM, Gonzalez B, et al. Parkinson's Disease Progression and Exposure to Contaminated Water at Camp Lejeune. *Mov Disord*. 2024;39(10):1732-1739.

[redacted]

TCE and PCE: Summary Discussion

In 1980, drinking water contaminants, primarily trichloroethylene (TCE) and perchloroethylene (PCE), were discovered at Camp Lejeune. A 2015 Institute of Medicine review of Veterans Affairs Clinical Guidance concluded "…Parkinson disease is a neurobehavioral effect that may result from exposure to TCE and/or PCE."[132] In 2017, following the Honoring America's Veterans and Caring for Camp Lejeune Families Act of 2012 (Camp Lejeune Act), the Department of Veterans Affairs established a presumptive service connection for anyone who served at Camp Lejeune for 30 days or more between August 1, 1953, and December 31, 1987 and later developed Parkinson disease.[133]

In December 2024, the United States government announced that the "Environmental Protection Agency (EPA) finalized the latest risk management rules for trichloroethylene (TCE) and perchloroethylene (PCE) under the bipartisan 2016 Toxic Substances Control Act (TSCA) amendments, marking another major milestone for chemical safety after decades of inadequate protections and serious delays."[134] The EPA report states that "PCE's hazards are well established." "The most sensitive health effect driving the unreasonable risk of PCE and selected as the basis for this rule is neurotoxicity from chronic exposure. It was selected based on the best available science and weight of scientific evidence and in consideration of the severity of the hazards, magnitude of exposure, population exposed, and uncertainties in the December 2020 Risk Evaluation for PCE and December 2022 revised risk determination for PCE." "EPA identified neurotoxicity as the most robust and sensitive endpoint for non-cancer adverse effects from acute inhalation and

[132] Committee on the Review of Clinical Guidance for the Care of Health Conditions Identified by the Camp Lejeune Legislation; Board on the Health of Select Populations; Institute of Medicine. *Review of VA Clinical Guidance for the Health Conditions Identified by the Camp Lejeune Legislation*. Washington (DC): National Academies Press (US); March 26, 2015.

[133] Department of Veterans Affairs. Diseases associated with exposure to contaminants in the water supply at Camp Lejeune, Final Rule. *Fed Reg*. 2017;82:4173-4185.

[134] Biden-Harris Administration Announces Latest Actions under Nation's Chemical Safety Law to Protect People from Cancer-Causing Chemicals Trichloroethylene and Perchloroethylene.

dermal exposures and as the most robust and sensitive endpoint for non-cancer adverse effects from chronic inhalation and dermal exposures for all conditions of use."[135,136] The EPA also determined that TCE poses an unreasonable risk to human health. In the TCE risk characterization, EPA identified neurotoxicity as a significant risk from acute and chronic inhalation and dermal TCE exposure.[137] The final EPA rules ban all uses of TCE, most uses of PCE, require worker protections under the Toxic Substances Control Act, and provide for 50 years of ongoing cleanup at Superfund sites.

In Section V, I presented exposure charts provided to me from Plaintiff's expert Dr. Kelly Reynolds. Dr. Reynolds' charts support my opinions that Ms. Rothchild had substantial exposure to toxic chemicals at Camp Lejeune. The charts detail a reasonable, estimated consumption dose for Ms. Rothchild. Exposure to these levels of PCE alone represent a substantial exposure. However, what must be noted is that these charts only relate to the ingestion exposure for Ms. Rothchild. We know Ms. Rothchild was exposed to PCE and TCE chemicals in the water through inhalation and dermal exposure as well. While the numbers in this chart are indicative of a very significant and substantial exposure in and of themselves, these numbers are only a part of the full exposure we know Ms. Rothchild experienced during her time at Camp Lejeune.

At a PCE concentrations of 40.1 to 43.76 parts per billion (ppb), or µg/L, as estimated specifically for Ms. Rothchild during her time working at Camp Lejeune, her PCE exposure is far above the EPA's maximum contaminant levels in drinking water of 5 ppb.[138] Exposure to median PCE levels of 15.4 ppb is substantial and known to cause Parkinson's disease. Furthermore, Ms. Rothchild's time on Camp Lejeune far exceeded the 90 day requirement for inclusion in Goldman et al. 2023 and the 30 day inclusion

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[135] EPA. Perchloroethylene (PCE); Regulation Under the Toxic Substances Control Act (TSCA). Document ID No. EPA-HQ-OPPT-2020-0720. EPA Publication No. 89 FR 103560. December 2024. https://www.federalregister.gov/documents/2024/12/18/2024-30117/ perchloroethylene-pce-regulation-under-the-toxic-substances-control-act-tsca#h-20.

[136] EPA. Risk Evaluation for Perchloroethylene. Document ID No. EPA-HQ-OPPT-2020-0720-0035. EPA Publication No. 740-R1-8011. December 2020. https://www.regulations.gov/document/EPA-HQ-OPPT-2019-0502-0058.

[137] EPA. Final Revised Unreasonable Risk Determination for Trichloroethylene, January 2023. https://www.epa.gov/system/files/documents/2023-01/TCE_Final%20Revised%20RD_12-21-22-FINAL-v2.pdf.

[138] National primary drinking water regulations. Environmental Protection Agency; 2009. Accessed February 3, 2025. https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations#Organic

IX. Opinions

Based on my education, training, experience, expertise, review of Ms. Diane Rothchild's case, and review of the medical and scientific literature, I conclude to a reasonable degree of medical and scientific certainty that:

1. I have considered the expert reports of Drs. Boehme and Cannon, and I agree with their review of the literature, opinions, and the basis for their opinions. I have also conducted my own review and concluded that it is as least as likely as not that exposure to the water at Camp Lejeune, contaminated with PCE and TCE, can cause Parkinson Disease.

2. I agree with Dr. Perlmutter's diagnosis of Parkinson Disease for Ms. Rothchild, and it is my opinion, to a reasonable degree of scientific and medical certainty that Ms. Rothchild's exposure to the water at Camp Lejeune is at least as likely as not the cause of her Parkinson Disease.

Kristin Andruska, MD, PhD

February 6, 2025